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Equine Exertional Myopathy

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Exercise related myopathies of horses have been recognized by horse owners and veterinarians for more than 100 years.¹ Symptoms ranging from mild alterations in gait and attitude to severe muscle spasm, immobility, and myoglobinuria are common. Exertional myopathy has had many names: azoturia, Monday morning disease, tying-up, cording-up, paralytic myoglobinuria, paralytic hemoglobinuria, exertional rhabdomyolysis, and simply myositis, have been used to describe it in the literature. Many clinicians feel that azoturia and tying-up represent two different diseases with different etiologies.^{2,3,4,5} Most, however, have come to believe that there is one disease process, more appropriately termed exertional myopathy, with a wide range in severity of clinical signs.

The name is not the only area of controversy associated with the disease. There is very little about this syndrome that clinicians and researchers can agree on. In 1917, Steffin observed, "No one disease of horses has been the subject of so many theories, theoretical treatments, and hypothetical suggestions as this one."⁶ In the past 50 years very little has changed. The vast number of published ideas on the pathogenesis of this disease is only surpassed by the number of different treatments applied to it in the field.

The one thing most individuals familiar with the disease can agree on, is the need for more research. Similar syndromes have been documented in other animal species and man.^{1,7} Investigations in these species, as well as some recent studies of the disease in the horse, have begun to shed some light on the mechanisms involved and more appropriate treatments.

Exertional myopathy occurs in a wide spectrum of the horse population under many conditions. The majority of cases, however, occur in Standardbred and Thoroughbred race horses in training. Classically the condition is seen in horses which have experienced a training lay-off of one or more days while continuing to take in a high calorie grain ration. Young horses first entering training are thought to be more susceptible.^{4,8-10} Often these horses are overly fat following preparation for sales. Fat, poorly conditioned animals of any age appear to contract the condition more often than those who are well trained.^{3,4,8,11}

Many researchers have reported a higher percentage of mares and fillies affected when compared to their male counterparts.^{1,3,10-14} Churchill reported as much as 95% of the cases he saw on the track were mares.³ McLean suggests a familiar predisposition¹; while others consider temperamental highly nervous animals more at risk.^{3,4,15} Inclement weather,^{3,4,8,14} steroid therapy,¹⁶ heavy muscling,^{1,4,9} anesthesia,^{7,17} foaling,¹⁷ and even release to pasture in the spring⁷ have all been documented to result in an increased incidence of the disease.

Once an animal has experienced an attack of exertional myopathy, repeated bouts are quite common. Such animals must be managed very carefully.

Clinical Signs

The clinical signs may be quite varied and can occur shortly after the onset of exercise or up to an hour afterwards. Affected horses may show only a stiff, stilted gait of the hind limbs, or may become so painfully affected that movement of any kind becomes impossible. Forced exercise in severely affected animals may exacerbate the condition and result in recumbency and an inability to stand. A classical "dog sitting" stance may also be observed. The affected musculature is often extremely firm and very painful when palpated. Localized twitching or, in severe cases, regional muscle spasms are

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also seen. The large muscle groups of the hind limbs are most often affected although the fore limbs are not exempt. The condition has been reported to be both unilateral and bilateral.

Affected animals may appear quite anxious due to pain, and evaluated heart and respiratory rates (even panting) are common. Elevations in temperature as high as 105 degrees are possible. Workers in Japan observed a decrease in gastrointestinal motility consistently in the cases they have studied.¹² Generalized and localized sweating is a common feature and is thought by some to be related to the pathogenesis of the disease.^{5,14,18} Severe cases with the absence of sweating have been associated with an exhaustion syndrome in endurance horses, and is considered to be a poor prognostic sign.

The urine in horses with exertional myopathy may be normal to dark coffee colored due to myoglobin leaking out of damaged muscle cells. Myoglobinuria is present in nearly all severe cases and occurs variably in mild to moderate attacks. Myoglobin levels in the urine must reach 40 mg/dl before being visually detectable.¹

Differential Diagnosis

Many conditions can be confused clinically with exertional myopathy. Because signs primarily involve the hindquarters and back, conditions of the vertebral column must be differentiated including: arthritis, spondylitis, and neuritis. In these conditions, the muscles are usually not hard and sweating is not a common feature. Hypoxic myopathy of the rear limbs associated with iliac thrombosis in *Strongylus vulgaris* infections, must also be considered.¹³ In this condition pain is variable, and the primary feature is loss of function due to vascular compromise. Attacks occur during exercise. The hind limbs are cool and refill times of the saphenous veins are increased. Rectal palpation reveals a decreased pulse pressure and a thickening of the effected arteries. Another condition associated with exercise is acute muscle strain with resulting hemorrhage and tearing of fibers. These animals are extremely tender over the affected muscles and swelling, which is not seen in exertional myopathy, is nearly always observed. Because the source of pain may not be readily apparent, the differential diagnosis should also include laminitis, tetanus, pleuritis, and colic.

Laboratory Data

Most investigators have recognized an increase in serum enzyme levels associated with exertional myopathy. Damage to cell membranes in the course of this disease result in leakage of cytoplasmic en-

zymes into the extracellular fluid and serum. The enzymes commonly evaluated include creatine phosphokinase (CPK), lactate dehydrogenase (LDH), aldolase (ALD), and aspartate aminotransferase (AST), formerly known as glutamic-oxaloacetic transaminase (SGOT). CPK and ALD because of their muscle specificity and ability to reveal small amounts of small muscle damage, are of great advantage early in the disease process. CPK rises quickly and peaks 6 hours following an attack. It declines over a period of 2 to 3 days. AST and LDH are not muscle specific, but increases in serum are seen with damage to other organs as well. They tend to peak at about 24 hours and recede slowly over 1 to 2 weeks, making them diagnostically valuable long after the acute phase of the disease is over. Levels of all the enzymes correlate well with the degree of muscle damage if other causes for AST and LDH elevations are eliminated.

Interpretation of all enzyme levels must be done in light of the animal's history. Exercise alone, with no muscle damage, has been shown to increase levels from 2 to 5 fold in horses which are poorly conditioned.^{19,20} Two fold increases are not uncommon in well conditioned horses following their normal training workouts.²⁹ Elevations of more than 20 fold in the serum of horses with severe muscle damage are routinely seen.^{7,13,20}

Acidosis has long been thought to be a feature of exertional myopathy. Exercising horses above the anaerobic threshold (speeds greater than 600 meters/min) results in a transient acidosis which is rapidly resolved during rest, usually within 60 minutes.^{8,13,21} A persistent acidosis in horses with exertional myopathy has not been substantiated. The one case cited in the literature involved a draft horse with severe muscle necrosis and a resultant hyperkalemia. The total serum CO₂ in this horse remained normal.³ Normal acid-base status, or more often a metabolic alkalosis, has been reported by most investigators.^{11,13,21,22} Establishing an animal's acid-base parameters prior to fluid therapy is imperative with this disease.

The most common electrolyte abnormalities found with the syndrome were hypochloremia and hypokalemia.^{8,11,13,18} Sweat has high concentrations of these electrolytes, and often profuse sweating is found in horses with exertional myopathy. This is thought to be a major source of potassium and chloride loss. For every 100 mEq of sodium, approximately 130 mEq of chloride and 40 mEq of potassium are lost in the sweat.¹⁸ Significant amounts of calcium and magnesium may be lost in this manner as well, accounting for the low values of these electrolytes reported by some.^{8,21,22}

Muscle biopsies of horses affected with exertional myopathy may be quite useful as a diagnostic and prognostic tool. Both surgical and fine needle techniques can be used for this purpose. Serial samples may be used to follow the progression, and estimate the probability of resolution of muscle damage. On histochemical evaluation, investigators have found depletion of glycogen, ATP, and creatine phosphate.³ Lindholm reported high muscle lactate levels in only four of twelve horses biopsied within 1 to 4 hours of an attack. The other 8 horses, and all samples taken after 4 hours were normal.¹⁶ Histopathologically myofibrillar necrosis, hyaline degeneration and variable inflammatory infiltration are seen.^{1,3,7,13,16}

Because the myocardium is not exempt from the metabolic and myopathic changes of the syndrome, ECG evaluation is appropriate for all horses afflicted with exertional myopathy. Grodski reported ECG abnormalities in 85% of the horses he studied.¹ Histopathologic evidence of myocarditis has also been seen.^{1,7}

Because myoglobin leakage is a consistent feature of damaged muscle cells, measurements of serum and especially urine levels are commonly done. Watanabe et.al. established that myoglobin levels in serum as measured by immunodiffusion were proportional to the severity of muscle damage and were useful early in the disease for determining prognosis. No visual discoloration of the serum is expected because of the low renal threshold for myoglobin.⁷ Urine myoglobin may be measured quickly using orthotolidine reagent strips, or more accurately by electrophoresis and spectrophotometry.

Because of the possibility of severe kidney damage due to myoglobin precipitation in the renal tubules, BUN and serum creatinine levels should be measured on all horses with myoglobinuria.

Pathophysiology

The mechanisms behind the muscle damage in exertional myopathy have long been the subject of speculation. A widely accepted pathogenesis was proposed by Carlstrom in 1931.²³ He postulated that during the rest period associated with many attacks, the muscle cells store excess glycogen. When the horse is subsequently exercised anaerobic glycolysis results in the accumulation of lactic acid. If the blood supply to the affected muscle cannot remove the acid rapidly enough, cellular necrosis occurs. Because many horses afflicted with exertional myopathy are not exercised at speeds above the anaerobic threshold, the reasoning behind the glycogen breakdown is difficult to explain. Some

researchers feel another cause must be found. Defective metabolism, enzyme abnormalities, thiamine deficiency, hypocalcemia, and potassium deficiency at the muscle level, have all been set forth as possibilities. The latter appears most probable.

Normal contraction of muscle cells results in the release of potassium to the extracellular fluid. This is thought to be the primary mechanism for increasing blood flow to the working muscles.^{5,14,18} If muscle potassium is low, this does not occur and a decreased perfusion is seen. Decreased perfusion results in decreased oxygenation and the need for anaerobic utilization of glycogen. There is also a decrease in waste clearance and thus a mechanism for lactic acid accumulation in the muscle. Because poor blood flow slows release of the acid into circulation, a systemic acidosis is not expected.

Therapy

Because the exact mechanisms behind the disease are still a matter of speculation, treatment can be little more than symptomatic. Attempts by individuals to correct theoretical, unproven deficiencies and imbalances have resulted in a vast number of regimes of questionable value. When presented with an animal in the acute phases of exertional myopathy the practitioner must assess the animal's condition and apply sensible, appropriate treatments for the symptoms at hand.

At the onset of an attack, exercise should be discontinued immediately. Continued activity, especially in severe cases, can result in worsening of clinical signs and a poorer prognosis. If the animal is only mildly affected he may be blanketed and walked to an appropriate place for treatment. In very severe cases, the animal may need to be treated on the spot and allowed to stay there or be trailered to the stable.

Assessment of the acid-base and electrolyte balances as a basis for fluid therapy is of great importance. Because of the complexity of the disease, these vary a great deal from one case to another. Appropriate fluid replacement should be administered as soon as these values are available. If the animal needs emergency fluid support, or if laboratory facilities are not available, a balanced electrolyte solution such as lactated Ringer's may be used.

If the animal is in a great deal of pain or showing signs of anxiety, tranquilization may be of some benefit. Acetylpromazine maleate or other phenothiazine derivatives have been used successfully. Care must be taken to replace all fluid deficits prior to using these because of the alpha blockade they

can produce. The vasodilation produced is thought to aid in restoring adequate perfusion to the damaged muscles when fluid volumes are adequate.

Nonsteroidal antiinflammatory drugs such as phenylbutazone and flunixin meglumine are recommended for both their antiinflammatory and analgesic effects.

Corticosteroids may be indicated early in the disease because of their stabilizing effect on cell membranes. This should help prevent further muscle damage. They also produce a relaxation of capillary sphincters improving perfusion somewhat. Muscle relaxants such as methocarbamol have recently been tried with some success. At dose rates of 15-25 mg/kg IV reduction of pain and relief of muscle spasm may be seen.¹¹

In the event of myoglobinuria, fluid therapy sufficient to result in diuresis is indicated in an effort to prevent tubular damage caused by pigment precipitation. Stronger diuretics are contraindicated due to the possibility of exacerbating an already present electrolyte imbalance and compromised circulatory system.

Close observation and good nursing care are essential to successful treatment of the initial insult. Occasionally, it becomes necessary to catheterize an animal to relieve stranguria and urine retention. In recumbent animals, soft deep bedding and frequent turning should be provided to prevent decubitus ulcers. Animals affected seriously enough to result in prolonged recumbency represent a poor prognosis.

Complications

Severe cases of exertional myopathy can have many outcomes. The animal may die due to renal failure, cardiac arrhythmias secondary to electrolyte imbalances or septicemia if recumbant. Direct myocardial involvement may lead to fatal cardiac dysfunction. If the animal survives the acute stages of the disease atrophy and fibrosis may occur in the effected muscles. Affected animals seem to be more prone to repeated bouts of the disease.

Prevention

Many modes of therapy have been used in an effort to prevent the recurrence of exertional myopathy in susceptible horses. Vitamin E and selenium supplements have long been advocated in prophylaxis. Their efficacy is questionable. Oral Nabcarbonate and dantrolene Na have been used with some success.¹¹

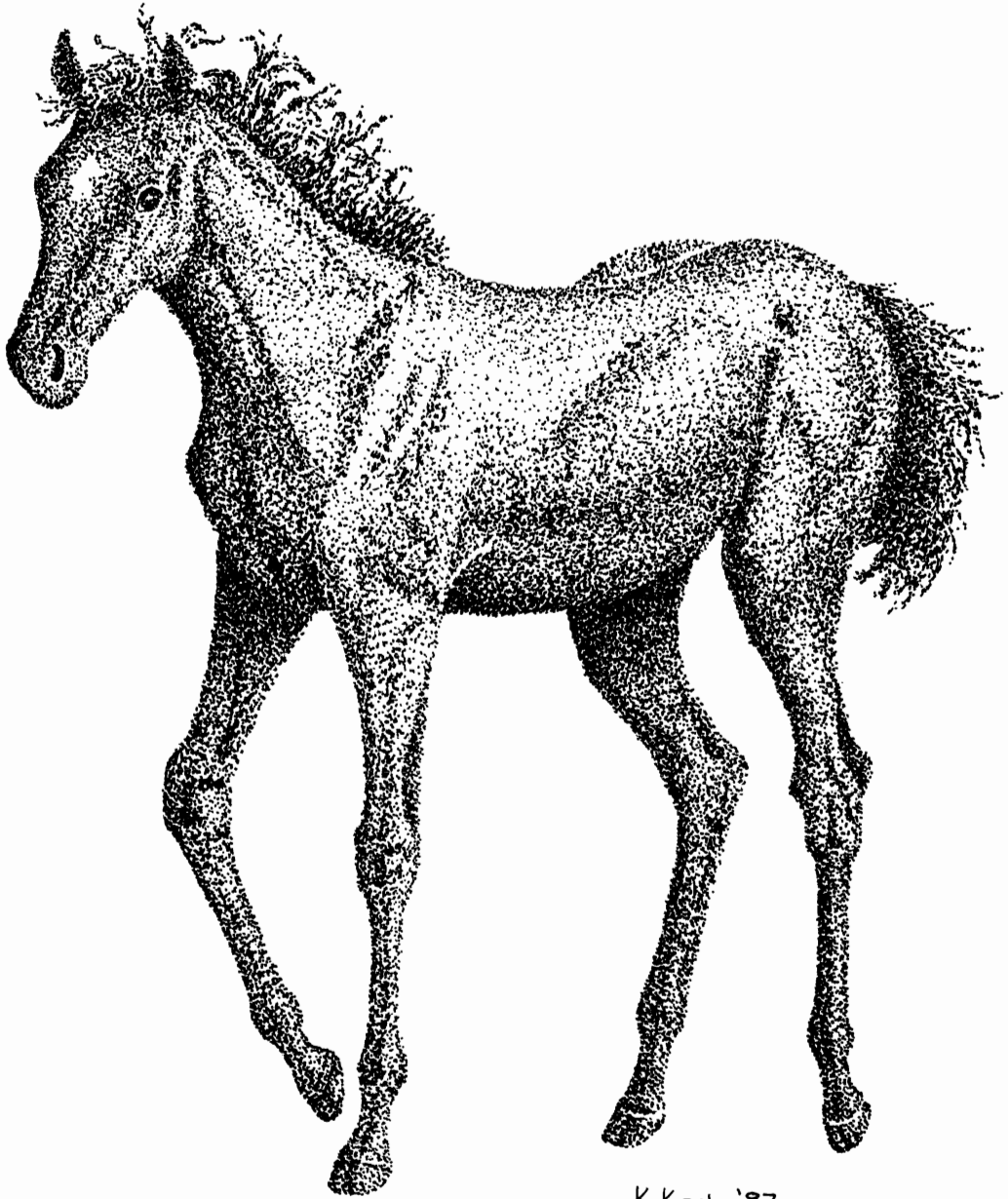
Management still remains the most important

factor in preventing recurrence. A consistent feeding and training schedule is imperative. If the animal is to be laid off for a day or longer, grain should be cut back or withheld. The exercise program should vary as little as possible. Some horses are so susceptible that they are not suitable for the work they are doing and may need to be simply transferred to an alternate career. The possibility of a heritable predisposition to this disease has not been ruled out. Therefore, whether these animals should be used as breeding stock is questionable.

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